CONUS MEDULLARIS SYNDROME DUE TO EPIDURAL ABSCESS BY STREPTOCOCCUS ORALIS AFTER STEROID INJECTION FOR SPINAL STENOSIS

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ABSTRACT

A spinal abscess is an uncommon but critical complication from spinal steroids injections. Unless there are contraindications to its use, gadolinium-enhanced Magnetic Resonance Imaging is the study of choice for detecting spinal epidural abscesses. As expected, patients with neurologic deficits are more likely to undergo urgent surgery and patients presenting with pain alone were more likely to be treated conservatively.

Our clinical case-report suggests that improved outcomes in spinal epidural abscess are more readily achieved via urgent surgery rather than with a conservative non-operative approach. Streptococcus oralis is a part of the normal human oral microbiota, capable of opportunistic pathogenicity. In this case, the vector hypothesis points towards an exogenous source of bacteria. It´s recommended that maximal barrier precautions consisting of full hand washing and the wearing of a sterile gown and gloves, a cap, face mask and the use of a large sterile drape should be instituted when conducting invasive spinal procedures.

KEYWORDS: spinal stenosis, lumbar steroid injection, epidural abscess, Streptococcus oralis.

Introduction

Spinal stenosis is one of the most common causes of either chronic back and leg pain or intermittent claudication in older people. After the failure of conservative treatment, nonsurgical modalities such as epidural steroid injections are contemplated to improve patient´s quality of life (Weinstein et al, 2008). Epidural hematoma or abscess is an infrequent and rare complication, but its clinical implication requires a rapid diagnosis and treatment due to long-term severe adverse effects (Haig and Tomkins, 2010). We present a case report describing the management of an epidural abscess produced by Streptococcus oralis after a steroid injection for the treatment of a severe spinal stenosis. It was finally successfully resolved after surgical drain by the neurosurgery team. The main purpose of this clinic case is to prevent this grave complication by using sterile conditions when performing any neuraxial pain procedure. International Education and Research Journal can be the way to distribute this knowledge among the scientific community.

Materials and Methods:

We present an 82-years old man, who was under treatment in the Chronic Pain Unit due to a central severe lumbar multilevel (from 2nd lumbar vertebra [L2] to 5th lumbar vertebra [L5]), and also, L5 left severe foraminal spinal stenosis. No early medical diseases were present except high blood pressure and a short brain stroke. The pain treatment was based on lumbar interlaminar epidural steroid injections, performed with 12 mg betamethasone in saline 6 mL, each 6 months. 24 hours after the last injection on August 2014, at the L2-L3 lumbar level, he presented a significant pain increase at the lumbar region that was treated with 1st and 2nd line oral analgesics (metamizole, acetaminophen and tramadol) with an inadequate response. He was also taking 100 mg acetylsalicylic acid daily that was immediately removed. He had no history of either recent dental work or oral surgery. Seven days later, due to severe persistent pain, he was hospitalized in the neurosurgery unit. The contrast-enhanced Magnetic Resonance Imaging (MRI) (image nº1) detected an image compatible with an epidural hematoma from 12th thoracic vertebra (T12) to L2 (maximum width 6 mm at L1-L2 level).

Fever was not present, and neurologic examination was unremarkable. Indeed, the patient initiated neurological symptoms 4 days later with urination difficulty and increased pain irradiated to either perineum or both legs with gait impairment. Urinary catheterization was needed, and a new MRI was performed showing an increase in the previously described image (maximum width 9 mm) and conus *medullaris* compression (image nº2).

Image nº1

The Magnetic Resonance Imaging (MRI) detected an image compatible with an epidural hematoma from T12 to L2 (red flags). The black and white flag shows the epidural injection level at L2-L3.

Image nº 2

Differential diagnosis included epidural hematoma, epidural abscess or therapeutic injection fluid mass. The surgical team indicated an urgent surgery, and a T12- L1 laminectomy was performed under general anesthesia. An epidural abscess was drained. Gram stain performed directly on the collection sample showed the presence of abundant leukocytes and numerous gram-positive short chains of cocci. After a 48 h incubation period, alpha-hemolytic colonies of streptococci grew on blood agar plates. The isolated strain was identified as Streptococcus oralis by using the matrix-assisted laser desorption ionization time of flight mass spectrometry (MALDI-TOF-MS) ™ (Bruker Daltonik GmbH, Bremen, Germany). Antimicrobial susceptibility testing was performed by broth microdilution method, using the Clinical Laboratory Standard Institute interpretative criteria (Clinical and Laboratory Standards Institute, 2015). The following minimal inhibitory concentrations (MIC) values were obtained: Penicillin <0.03 mg/L; amoxicillin: <0.5 mg/L; ceftriaxone: 0.12 mg/L; erythromycin <0.25 mg/L; clindamycin: <0.25 mg/L; levofloxacin 2 mg/L; imipenem <0.12 mg/L; vancomycin <0.5 mg/L; trimethoprim-sulfamethoxazole <0.5/9.5 mg/L; rifampicin: <0.25 mg/L. After surgical drain, empiric antimicrobial therapy was started with piperacillin-tazobactam IV infusion (2/0.5 g every 6h) during 48h. Ceftriaxone IV 2g/24h during 10 days was continued, and finally, amoxicillin orally 2g/8h during one month was administrated. Patient´s clinical evolution was satisfactory due to rehabilitation from both urine retention and walking difficulty. Six months later, the patient presented a complete recovery from the acute neurological symptoms, remaining those related to the lumbar spinal stenosis. A control MRI was made, and only minimum points of myelopathy at L-1 level were observed. The patient refused to repeat any other epidural injection.

Discussion:
Lumbar spinal stenosis is defined as a narrowing of the spinal canal, secondary to several causes such as disc herniation, protrusion, extrusion, and disc bulging combined with osteophytes and arthritic changes of the facet joints. Clinical signs are caused by entrapment and compression of the intraspinal, vascular and nervous structures. Thus, symptoms of spinal stenosis may be related to a neurovascular mechanism such as nerve root excitation either by local inflammation or compression of the central canal arterial, low flow in cauda equina, venous congestion or increased epidural pressure (Kalichman et al, 2009). While surgery is the most common treatment for lumbar spinal stenosis, epidural injections are common nonsurgical interventions. The primary objective of surgery is to decompress the thecal sac from the spinal canal compromise. Otherwise, the goal of epidural injections is to suppress pain response and improve function by an anti-inflammatory effect. (de Schepper, 2103) (Bae et al, 2013)

The effectiveness of epidural injections in the management of lumbar central spinal stenosis has been under debate along past years. Many authors have reported a lack of efficacy of epidural injections in the management of chronic pain and disability in comparison with the gold standard status of surgical intervention (Kovacs et al, 2011). However, various systematic reviews have demonstrated clinical efficacy and cost effectiveness of epidural injections in managing pain

due to spinal stenosis (Bicket et al, 2013). Moreover, based on one recent systematic review, the evidence is Level II [in a grade from I (the best) to V (the worst)] for either lumbar interlaminar or caudal epidural injections for lumbar spinal stenosis (Kaye et al, 2015). These support a good evidence level for the interventional procedure in our clinical case presentation.

Spinal epidural abscesses (SEAS) were once considered a rare form of spinal infection. They were typically found in people 60 to 70 years old with an estimated incidence of rates higher than 10 per 10,000 admissions. This rising incidence is likely a reflection of the aging population, a greater number of patients with predisposing medical conditions (older age and metabolic disease), and an increased societal prevalence of intravenous drug use and alcoholism. Other risk factors include recent spinal or epidural procedures, recent spinal trauma, Human Immunodeficiency Virus infection (HIV), and chronic steroid use. (Curry et al, 2005). Before antibiotics were widely available, SEAs were a fatal condition most commonly diagnosed at autopsy. Moreover, advances in health care have led to improved clinical diagnosis and outcomes. However, SEA remains a source of significant morbidity and mortality nowadays. A meta-analysis (Reihsaus et al, 2000) found that up to 34% of patients presented with paralysis and mortality rates were reported between 12% and 34%. The principal pathogen identified was S. aureus (SA), 73% determined in the meta-analysis including 915 patients. Overwhelmingly, studies highlight the importance of early diagnosis and urgent surgical intervention. Surgery is considered necessary in the setting of SEA when patients present with evolving neurologic deterioration, spinal instability, or persistence of infection despite antibiotic treatment. In the absence of these criteria, however, the necessity of surgery has been challenged (Leys, 1985). The authors, proposed multilevel abscesses, neurologically intact patients, or the presence of a complete spinal cord injury for more than 72 hours as relative indications for non-operative management. A more recent review (Darouiche, 2006) expanded these relative indications to include patient refusal for surgery and narrowed the window of preexisting paralysis to less than 24 to 36 hours. Despite reports of successful medical outcomes, failure of intravenous antibiotics and delayed surgical intervention often result in severe consequences. If medical management alone is to be considered, it is imperative to identify which group of patients is most amenable to non-operative treatment. Moreover, even in the absence of neurologic deficit involving the spinal cord, the presence of the other independent risk factors predicted a very high failure rate SEA treated with medical management alone has a very high risk of failure if the patient is older than 65 years with diabetes mellitus (DM), Methicillin-Resistant SA infection (MRSA), or neurologic compromise. In the absence of these risk factors, non-operative management of spinal epidural abscess may be considered as the initial line of treatment with culture-specific intravenous antibiotics and close monitoring by a spine surgeon (Kim et al, 2104). In our clinical description, the non-surgical treatment was the initial choice. When cauda equina symptoms appeared, 10 days after the epidural injection, urgent surgery was performed. Outcomes for compressive lesions (epidural hematoma or spinal epidural abscess) depend on the severity of neurologic impairment and the duration of symptoms at the time of neurosurgical decompression. Most experts agree that neurologic recovery is improved with early decompression (<8–12 h from symptom onset in epidural hematoma and <36 h from symptom onset for spinal epidural abscess) and when the preoperative neurologic deficits are milder in severity (Neal et al., 2105).

Streptococcus oralis is a member of the Streptococci Viridans Group (SVG), which is part of the human oral and pharyngeal microbiota. SVG are rarely cause of disease and their isolation in clinical samples obtained from non-sterile sites is often considered as contamination. However, their presence may be associated with endocarditis, bacteremia, septicemia, meningitis or pneumonia. The usual pathogenic mechanism is the endogenous source, as the result of blood invasion from oral cavity after dental or oropharyngeal surgical procedures. However, direct transmission of bacteria via respiratory droplets generated via coughing, sneezing or talking may also occur. SVG rarely colonize the skin, unlike other bacteria such as Staphylococci, Pseudomonas, Enterobacteriaceae, and Enterococci. SGV can rarely survive outside their natural habitat (oropharynx), so the acquisition of the bacteria across surfaces or solutions-contaminated liquids is outstanding.

In our case, the acquisition hypothesis points towards an exogenous source of bacteria Streptococcus oralis from the upper airway of practitioner pain team. In particular, droplet transmission from the upper airway is thought to result in direct inoculation of the organism into the tissues surrounding medullary canal by the spinal needle. Since the market inclusion of single-use devices, exogenous contaminated sources have become less involved. However, bacterial transmission from the physician as a result of poor aseptic technique has been discussed previously causing meningitis. This fact is supported by the clustering of cases of bacterial meningitis following spinal anesthesia (Rubin et al, 2007). The Association of Anesthetists of Great Britain and Ireland issued guidance in 2008 regarding infection control precautions for anesthetic procedures. The guidance describes the barrier precautions consisting of the use of a large sterile drape, full both hand washing and the wearing of a sterile gloves and gown, a face mask and a cap. Theses should always be instituted when conducting invasive spinal procedures (Association of Anaesthetists of Great Britain and Ireland, 2008). Although this guidance exists, published evidence surrounding wearing a face mask is controversial. Moreover, clinical evidence in healthy volunteers has con-

firmed that they are effective at reducing forward bacterial dispersion at 30 cm from the mouth. The general recommendation is that facemask may prove beneficial in these circumstances (Philips et al, 1992). In our case, sterile gloves were born both by the anesthesiologist and the nursery team during the spinal puncture. However, facial mask and cap were not used and this fact could be the key-point to improve general pain physician's medical education (Hebl, 2006).

Conclusions:
Most of the patients with SEA are associated with a disease (such as DM, HIV, Chronic renal failure, cancer, cirrhosis, etc.) or condition that suppress the immune system (e.g. alcoholism, IV drug abuse, trauma). Spinal injection is also a possible origin. The presenting symptoms are mainly spinal pain and fever. The neurological deficit might develop in hours or even in months due to compressive effect of epidural abscess or ischemia. To decrease morbidity rates, early diagnosis is mandatory. An MRI is considered standard of care once there is suspicion of SEA. The treatment mostly depends on early surgical decompression and prolonged antibiotic therapy. We agree that all patients presenting with neurologic deficit should undergo urgent surgical drainage. Conservative treatment should be reserved only for select population with significant comorbidities. Therefore, these patients should be closely followed by a neurosurgical service to detect neurologic deterioration.

The authors advocate a systematic approach to the assessment and early management of SEAs. Clinicians must keep in mind the possibility of iatrogenic spinal abscess following spinal steroid injection and the adherence to strict aseptic precautions to reduce the incidence of this serious complication.

FOOTNOTES:

- Image nº 2: MRI showing an increase in the maximum width from the hiperdensity T12-L2 image and conus medullaris compression (Black and white flag).

REFERENCES:

1. Association of Anaesthetists of Great Britain and Ireland. (2008). Infection control in anaesthesia. Anaesthesia, 63, pp. 1027-36.

2. Bae HW, Rajaee SS, Kanim LE. (2013). Nationwide trends in the surgical management of lumbar spinal stenosis", Spine, 38, pp. 916–26.

3. Bicket MC, Gupta A, Brown C, Cohen SP. (2013). Epidural injections for spinal pain: a systematic review and meta-analysis evaluating the "control" injections in randomized controlled trials. Anesthesiology, 119, pp. 907–31.

4. Clinical and Laboratory Standards Institute. (2015). Performance standards for antimicrobial susceptibility testing; Twenty-fifth informational supplement M100-S25. CLSI, Wayne, PA.

5. Curry WT, Hoh BL, Amin-Hanhani S, Eskandar EN, (2005). Spinal epidural abscess: clinical presentation, management, and outcome. Surgical Neurology, vol. 63, pp. 364-371.

6. Darouiche RO. (2006). Spinal epidural abscess. New England Journal of Medicine, 355, pp. 2012–2020.

7. de Schepper EI, Overdevest GM, Suri P, Peul WC, Oei EH, Koes BW, et al. (2013). Diagnosis of lumbar spinal stenosis: an updated systematic review of the accuracy of diagnostic tests, Spine, 38, pp. 469–81.

8. Haig AJ and Tomkins CC., (2010). Diagnosis and management of lumbar spinal stenosis. Journal of the American Medical Association, 303, pp. 71-72.

9. Hebl JR. (2006). The importance and implications of aseptic techniques during regional anesthesia. Regional Anesthesia & Pain Medicine, 31, pp. 311-23.

10. Kalichman L, Cole R, Kim DH, Li L, Suri P, Guermazi A, et al, (2009). Spinal stenosis prevalence and association with symptoms: the Framingham Study, Spine Journal, 9, pp. 545-550,

11. Kaye AD, L Manchikanti L, Abdi S, et al. (2015). Efficacy of Epidural Injections in Managing Chronic Spinal Pain: A Best Evidence Synthesis. Pain Physician, 18, pp. 939-1004.

12. Kim SD, Melikian R, Ju KL, Zurakowski D, Wood KB, Bono CM, et al., (2014).Independent predictors of failure of nonoperative management of spinal epidural abscesses. The Spine Journal, 14, pp. 1673–1679.

13. Kovacs FM, Urrutia G, Alarcon JD (2011). Surgery versus conservative treatment for symptomatic lumbar spinal stenosis: a systematic review of randomized controlled trials, Spine, 36, pp. 1335–1351.

14. Leys D, Lesoin F, Viaud C, Pasquier F, Rousseaux M, Jomin M, et al. (1985). Decreased morbidity from acute bacterial spinal epidural abscesses using computed tomography and nonsurgical treatment in selected patients. Annals of Neurology, 17, pp. 350–355.

15. Neal JM, Barrington MJ, Brull R, Hadzic A, Hebl JR, Horlocker TT, et al. (2015). The Second ASRA Practice Advisory on Neurologic Complications Associated With Regional Anesthesia and Pain Medicine: Executive Summary 2015. Regional Anesthesia and Pain Medicine, 40, pp. 401-30.

16. Philips BJ, Fergusson S, Armstrong P, Anderson FM, Wildsmith JA. (1992). Surgical face masks are effective in reducing bacterial contamination caused by dispersal from the upper airway. British Journal of Anaesthesia 69, pp. 407-408.

17. Reihsaus E, Waldbaur H, Seeling W. (2000), Spinal epidural abscess: a metaanalysis of 915 patients. Neurosurgical Review, 23, pp.175–204.

18. Rubin L, Sprecher H, Kabaha A, Weber G, Teitler N, Rishpon S. (2007). Meningitis following spinal anesthesia: 6 cases in 5 years. Infection Control & Hospital Epidemiology, 28, pp. 1187-1190.

19. Weinstein JN, Tosteson TD, Lurie JD, Tosteson AN, Blood E , Hanscom B, et al, (2008). Surgical versus nonsurgical therapy for lumbar spinal stenosis, New England Journal of Medicine, 358, pp. 794-810.